survivors than non-survivors is not surprising. For instance, in seven cases death followed withdrawal of mechanical ventilation. Was early tracheostomy practised? This increasingly is recognized to enable earlier discharge from the ICU.

It is difficult to evaluate quality of life in chronic illness. Dependency does not equate with reduced quality (4). A better evaluation would have been to interview survivors and ascertain whether they would opt for mechanical ventilation again. Other series (5), involving a much larger number of patients, suggest a better outcome than is reported here.

As with previous studies, analysis of prognostic indicators demonstrates that there are no sensitive measures and that an 'on the spot' decision, leaning towards provision of mechanical ventilation, is justified. NIV should be employed unless contra indications exist. If it fails to reverse the spiral towards intubation, it may be employed to speed weaning following a short period of MV. Surely this is a more appropriate way to care for our patients than the nihilistic one suggested by Dr Hill and colleagues.

DR A. C. DAVIDSON* AND
DR D. F. TREACHER†
*Consultant Physician,
Intensive Care and Respiratory Medicine
†Director of Intensive Care and Consultant Physician,
Guy's and St. Thomas' Hospital Trust,
London, U.K.

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Reply to Drs Davidson and Treacher

The letter by Davidson and Treacher suggests that the paper by Hill *et al.* (*Respir Med* 1998; **92:** 156–161) makes for depressing reading and suggests that other series show better outcomes. Our series revealed a 49% hospital mortality and 59% 1 year mortality: similar outcomes were found in U.S. and more recent U.K. studies. In the SUPPORT study (1) the 180 day mortality for 348 ventilated patients was 43% (1 year mortality not reported) but in another large U.S. study (2), 1 year follow-up was available on 167 ventilated COPD patients and in this series hospital mortality was 30%, 180 day mortality 48%, and 1 year mortality identical to our series at 59%. The largest U.K. series by Wildman *et al.* (3) of 242 ventilated COPD patients reports a hospital mortality of 34·2% and 180 day mortality of 35·5% (1 year mortality data not yet reported). We would not agree with the suggestion that the article will 'reinforce the prejudice in the U.K. against intubation and ventilation in acute exacerbations of COPD'. Davidson and Treacher seem to have missed the point of the study which was to stratify a complex case mix and identify factors that could prejudice poor outcome in 1993 when there was little discussion of this important topic in the U.K.

The low use of NIPPV in our series reflects evidence-based practice appropriate to the study period between 1993 and 1995. At that time the Bott paper (4) had been published suggesting the potential for NIPPV within clinical trials, but did not provide a secure evidence base for its wholesale adoption. The subsequent randomized controlled trials suggesting a role for NIPPV were not published until 1995 (5,6) and we agree with the two authors that the ICU management of COPD patients will not involve NIPPV, with data likely to emerge soon to help decide its role outside the ITU.

Davidson and Treacher question whether admission was delayed leading to an increase in mortality and whether early tracheostomy was practised in the series by Hill *et al.* The paper did look at whether being admitted to ITU on the day of hospital admission influenced outcome. In this study there was no difference in outcome whether being admitted directly to ITU or following deterioration with medical therapy from a medical ward. Tracheostomy was and is practised in our intensive care unit if there are no signs of ability to wean early and earlier still if tracheo-bronchial toilet is a problem.

Davidson and Treacher comment that lack of medical documentation should not be taken to indicate that treatment options were not discussed with patients or relatives, and question whether this would influence ICU admission. The courts would not necessarily agree with this; what is not recorded possibly has not been done — present views are not to give professionals the benefit of the doubt where medical records are concerned. Discussion can certainly influence decision making, for example, one would be more likely to ventilate someone with a good pre-morbid history without co-morbidities.

We would restate our contention that the high absolute and opportunity costs along with the significant morbidity and mortality in this group emphasizes the need for further prospective studies to better identify the patients who will benefit. We are presently initiating such a study at Birmingham Heartlands Hospital and preliminary results are expected later in 1999.

DR ADAM HILL,
DR MARTIN WILDMAN,
DR ROWLAND HOPKINSON* AND
DR DAVID STABLEFORTH
*Department of Respiratory Medicine and *Department of Anaesthetics, Birmingham Heartlands Hospital, Bordesley Green East, Birmingham B9 5ST, U.K.*

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Dear Editor

Corticosteroid treatment of asthma: now at the crossroads

I read with interest the observation of Seale and Donnelly (1) on the relative systemic activity of fluticasone proprionate (FP) and budesonide (BUD). This is based on studies in healthy volunteers. In a previous meta-analysis of studies performed in patients with asthma (2), I noted that if anything the effect was the opposite of the one they describe.

In order to address this apparent contradiction, I performed an analysis to consolidate the large volume of literature comparing the systematic effects on the HPA axis of FP versus BUD in healthy volunteers and asthmatics. To test the hypothesis that observations of systemic effects in volunteers may not predict the outcome in patients, I carried out a review of all studies of FP and BUD published to date which measured effects on cortisol (3–25). To avoid bias, data were selected only where both FP and BUD were compared in the same study. Use of plasma and urine samples and measurements of both AUC cortisol and AM cortisol, compared either to baseline or placebo, were reported. Where multiple measures were reported in a study, urine or plasma area under the curve and change from placebo was selected in preference to the less sensitive morning plasma cortisol data.

For the purpose of the analysis, the effect of any dose of FP or BUD on cortisol levels was assessed by using the residual level of cortisol remaining at the end of treatment expressed as a percentage of baseline (or placebo response). BUD and FP were then compared within each study using the ratio of the respective residual percentages; ratios greater than 1 indicate FP suppression greater than BUD. Data were used only from the highest dose pair of FP and BUD in any study. Table 1 shows the resulting doses compared and effect on cortisol levels together with their weighted (by group size) means.

At approximately equal doses, these results confirm that in healthy volunteers, FP gives rise to higher levels of cortisol suppression than BUD (BUD/FP ratio = 3·3). However in asthmatic patients, FP and BUD result in equal effects on the HPA axis (BUD/FP ratio = 1·0).

These data suggest a difference in the relative systemic exposure of healthy volunteers and asthmatic patients to FP and BUD. This is consistent with pharmacokinetic data which have shown that volunteers have two-fold higher FP levels than patients with asthma (26). From these pharmacokinetic data, one could infer a reduction in lung absorption in asthmatic patients which, given the negligible oral bioavailability of FP, would lead to a low systemic exposure to FP but not BUD as was observed in the current analysis. I conclude that studies of systemic exposure in healthy volunteers may not reflect the clinical outcome in patients. However, specifically designed studies should be used to test this hypothesis.

C. HALLETT, B.SC., CSTAT.
Consultant Statistician to GlaxoWellcome R and D., 14, Boleyn Avenue, Ewell, Epsom, Surrey, KT17 2QH, U.K.

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